Conferences and Reviews

Sinusitis

A Review for Generalists

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A frequent complication of the common cold, sinusitis is one of the most prevalent problems seen in general medical and emergency department practices. In addition, nosocomial sinus infection, particularly in intensive care units, is being recognized more frequently. Decision making about managing patients with sinusitis is based primarily on the history and, to a lesser extent, the findings of the physical examination.

(Reuler JB, Lucas LM, Kumar KL: Sinusitis—A review for generalists. West J Med 1995; 163:40-48)

Sinusitis is one of the most prevalent problems encountered in general medical practice. Acute bacterial sinusitis, the most frequent complication of the common cold, may be unrecognized or misdiagnosed. Some patients are predisposed to recurrent bouts of acute sinusitis, and chronic sinusitis may develop that presents as enigmatic facial or head pain. Finally, nosocomial sinusitis is being increasingly recognized in patients being treated in a hospital.

In most cases of sinusitis, clinical decision making rests on the history and physical findings. We review those aspects of sinusitis germane to general medical practice, including clinical and bacteriologic features, pathogenesis, special populations, treatment, and indications for radiographic imaging and surgical referral.*

Anatomy of the Sinuses

The accessory sinuses or air cells of the nose are called the paranasal sinuses. The frontal, ethmoid, sphenoid, and maxillary sinuses are lined with ciliated mucous membranes and are directly contiguous with the nasal cavity through the meatus of the superior, middle, and inferior nasal conchae arising from the lateral nasal walls (Figure 1). The maxillary sinus is inferior to the bony orbit and superior to the hard palate and has an ostium located superiorly and medially in the sinus, a location that impedes gravitational drainage.[1]

The ostium of the frontal sinus communicates with the nasal chamber at the frontal recess beneath the frontal sinus just ventral and superior to the infundibulum. The sphenoid sinus is just anterior to the pituitary fossa behind the posterior ethmoid sinuses, and its paired ostia drain into the sphenoethmoidal recess. Finally, the ethmoid sinuses comprise 3 to 15 air cells on each side, with each cell maintaining a separate drainage path to the nasal chamber. The lateral surface of this labyrinth forms the medial surface of the orbit.

The osteomeatal complex comprises the area between the middle and inferior turbinates at the confluence of the drainage of the frontal, ethmoid, and maxillary sinuses (see Figure 1). This area, where two mucosal layers come into contact, is predisposed to localized inflammatory change because of a disruption of mucociliary clearance, a resultant retention of secretions, and decreased sinus ventilation.

Pathogenesis

Four interrelated factors operate in the causation of sinusitis, including patency of the ostia, nasal airflow, mucociliary activity, and the nature and quantity of secretions. The sinus mucosa is similar to that in the respiratory tract and nasal passages. A mucous blanket is propelled toward the ostia by the underlying cilia, removing microorganisms, pollutants, and irritants. The obstruction of mucociliary drainage in the osteomeatal complex is thought to be a major cause of symptomatic sinus disease. Lower oxygen content in the sinuses facilitates the growth of organisms, impairs local defenses, and alters leukocyte function.[2] The swelling of the mucous membranes narrows the ostia and impairs the transport capacity of the mucociliary apparatus.

Viral upper respiratory tract infections and allergic rhinitis are the most common initiating factors in sinusitis.[3,4] Also, nasal polyps may cause sinusitis by impeding drainage and aeration of the antrum. Conversely, constant irritation from sinus infection may stimulate the growth of polyps by causing the nasal mucosa to duplicate in the form of a polyp. Barotrauma, as seen in airline pilots, may

*See also the editorial by J. W. Williams Jr, MD, "Sinusitis—Beginning a New Age of Enlightenment?" on pages 80-82 of this issue.

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ABBREVIATIONS USED IN TEXT

CT = computed tomography
HCl = hydrochloride
OR = odds ratio
VA = [Department of] Veterans Affairs

result in barometric pressure stresses leading to altered sinus drainage, impaired ventilation, and the clearance of mucous secretions predisposing to acute sinusitis.[5] Swimming, diving, and abuse of topical nasal decongestants, as well as the presence of hypertrophied adenoids, deviated nasal septum, or nasal foreign body, may predispose to obstruction of the osteomeatal complex, as can the placement of nasotracheal or nasogastric tubes.[6] Finally, contiguous dental infection was the cause of maxillary sinusitis in 10% of cases reported from an otolaryngology referral practice.[7]

Bacteriology

The paranasal sinuses are normally sterile. In adults, acute sinusitis is most commonly due to *Haemophilus influenzae* or *Streptococcus pneumoniae*.[4,8] In chronic sinusitis, anaerobic organisms, predominated by species of *Peptostreptococcus*, *Fusobacterium*, and *Prevotella* (formerly *Bacteroides*), are found in 88% of cultures, alone or in conjunction with aerobes, most commonly *Streptococcus* species or *Staphylococcus aureus*.[9-11] In those with underlying immune deficiencies, such as patients positive for the human immunodeficiency virus, organisms identified may include gram-negative species and opportunistic fungi.[12,13]

In patients with acute and chronic sinusitis, the presence or type of bacteria cannot be adequately determined by the use of nasal swabs.[3] Therefore, when accurate characterization of bacteria is warranted, either direct sinus aspiration or culture material obtained at the time of an operation is necessary.

Clinical Features

Acute Sinusitis

The symptoms of acute sinusitis, including facial pain, headache, purulent nasal discharge, decreased sense of smell, and fever, are nonspecific and often difficult to differentiate from those of the common cold and allergic rhinitis.[14,15] Most studies comparing the history and physical examination with the standard procedure, antral puncture, for diagnosing maxillary sinusitis have been conducted in otolaryngology practices.[7,16,17] In these groups, subjective or objective purulent nasal discharge was most helpful in differentiating sinusitis from rhinitis. Antral puncture, however, is not a practical diagnostic procedure for primary care providers.

A recent study of primary care patients compared general practitioners' clinical diagnosis of maxillary sinusitis with the use of ultrasonography of the sinuses (a technique used primarily in Europe).[14] Using an algorithm of five weighted symptoms—preceding common cold, purulent rhinorrhea, pain on bending, unilateral maxillary pain, and pain in the teeth—the proportion of correct diagnoses increased from 40% to 55%. Diagnosis with the algorithm was more accurate than clinical diagnosis by general practitioners, but inaccuracy and uncertainty persisted.

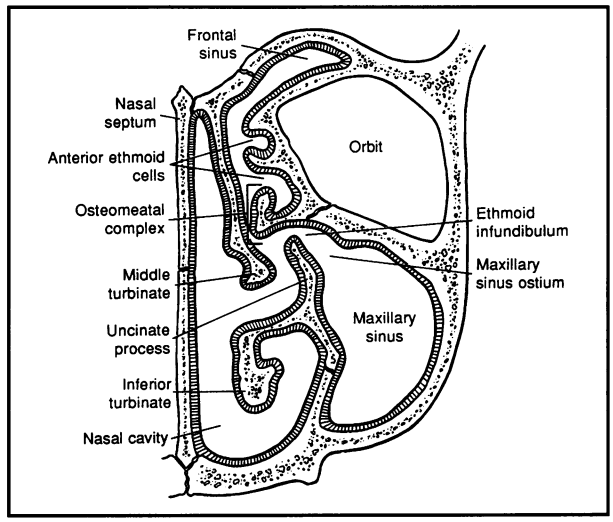

Figure 1.—The diagram shows the paranasal sinuses.

Finally, a recent study done in a general medicine clinic of a VA medical center evaluated 247 men (median age, 50 years) presenting with rhinorrhea, facial pain, or self-suspected sinusitis.[18] The prevalence of sinusitis in this group, as defined by radiographic changes, was 38%. Logistic regression analysis showed five independent predictors of sinusitis, none of which were both sensitive and specific alone: maxillary toothache (odds ratio [OR], 2.9), transillumination (OR, 2.7), poor response to nasal decongestant or antihistamine therapy (OR, 2.4), colored nasal discharge noted by the patient (OR, 2.2), or mucopurulence seen during examination (OR, 2.9); used in combination, the predictive probability varied from 92% (all 5 features present) to 9% (none present). An important finding is that the overall clinical impression was more accurate than any single historical or examination finding, suggesting that the gestalt is useful to diagnose sinusitis when the probability of disease is judged to be high, and antecedent upper respiratory tract infection or facial pain were not predictive of sinusitis. Whether these findings are applicable to other groups (such as younger patients) or settings (clinicians without a special interest in sinus disease or access to or training with special transillumination instruments) is not known.

Sinusitis and Immunosuppression

The clinical presentation of sinusitis in immunocompromised patients, particularly those with severe leukopenia, may be subtle, making an early diagnosis difficult and increasing the likelihood of a fulminant and even fatal course.[19] The common underlying diseases include aplastic anemia, leukemia, bone marrow transplantation, and primary immunodeficiency diseases. Besides the

common sinus pathogens, atypical mycobacteria, *Aspergillus* species, Phycomycetes, *Fusarium* species, and cytomegalovirus have been reported to cause sinusitis in these patients.[19]

Sinusitis occurs in patients with severe leukopenia and may present as fever of unknown cause, rhinorrhea, or facial edema. A lesser degree of pain, edema, erythema, and pus formation is due to an impaired inflammatory response. The physical examination may show pale or gray areas in the nasal mucosa that are the earliest signs of tissue infarction due to a fungal invasion of blood vessels. Rather than purulent, the discharge is often turbid.

Of particular concern is the invasive form of fungal sinusitis that may extend across the mucosa into bone and adjacent structures, including the orbits, brain, and cavernous sinus.[20] The diagnosis of invasive infection can be made only after the histologic demonstration of hyphae. Early diagnosis and extensive surgical debridement are crucial for survival.

Rhinocerebral mucormycosis is a fulminant infection similar to invasive aspergillosis that occurs in patients with diabetic ketoacidosis or acute leukemia. The infection originates in the nasal cavity and spreads through the sinuses to the orbit and central nervous system, causing thrombosis and tissue necrosis. Patients exhibit a black nasal mucous membrane, cranial nerve palsies, proptosis, and an altered mental state. Treatment involves emergency aggressive surgical debridement and the administration of parenteral amphotericin B.[21]

Hospital-Acquired Sinusitis

Hospital-acquired acute sinusitis is due to a different microbial flora than community-acquired acute sinusitis. *Staphylococcal aureus*, *Pseudomonas* species, *Klebsiella* species, and other gram-negative organisms are found in sinus puncture studies.[22-24] About 35% to 40% of cases of hospital-acquired sinusitis are polymicrobial infections and are often bilateral. The incidence of sinusitis in hospitalized patients requiring long-term ventilation varies widely depending on the definition of sinusitis (radiologic versus culture) and the patient population. Two recent studies using a combination of clinical signs, radiologic diagnosis, and aspirate confirmation found that, after two to four days of ventilation, those patients with nasotracheal intubation had a 0% to 40% greater incidence of nosocomial sinusitis compared with patients with orotracheal intubation.[6,24] Other predisposing factors for sinusitis in inpatients include nasogastric tubes, nasal packing, cranial and facial fractures, and the use of corticosteroids and of antibiotics.[22,25] Unexplained fever or leukocytosis and purulent nasal discharge may be the major clinical features that lead to the recognition of acute sinusitis in an intubated patient.

Allergic Sinusitis

Allergic sinusitis occurs in conjunction with allergic rhinitis. The mucosal changes in the sinuses are the same as seen in patients with allergic rhinitis. Symptoms usually consist of stuffiness, itching and burning of the nose, frequent bouts of sneezing, recurrent frontal headache, and a thin nasal discharge. Headache that is located between the eyes or in the frontal area is a common symptom and results from edema and swelling of the soft tissues. Allergic rhinitis may lead to polyp formation, and similar polypoid changes in the mucosa of the sinuses are seen commonly in patients with allergic sinusitis.

Although the term allergic sinusitis is commonly used, sinus disease associated with severe allergic rhinitis has never been documented radiographically. Single photon-emission computed tomography has been used to assess the metabolic activity and dynamic physiology of the sinuses.[26] Three patients with ragweed allergic rhinitis had a strongly positive skin reaction, were markedly symptomatic, their nasal mucosa was edematous, and their sinus x-ray films were normal. Single photon-emission computed tomographic imaging demonstrated substantial hyperemia of the sinuses and increased uptake in bones around the sinuses, which resolved after the ragweed season ended. This study shows that the dynamic process in the paranasal sinuses is similar to that seen in the nasal mucosa of allergic patients.

Chronic Sinusitis

Acute suppurative sinusitis is any bacterial infection in a paranasal sinus that lasts from one day to three weeks. When the infection lasts from three weeks to about three months, it is diagnosed as subacute sinusitis. A symptom complex of purulent nasal discharge, nasal obstruction, facial pain, headaches, chronic cough, and halitosis persisting longer than three months is labeled chronic sinusitis.[27] During the acute and subacute stages, the epithelial damage in the sinus is usually reversible, whereas in chronic sinusitis the mucosal damage is frequently irreversible, often requiring surgical therapy for better drainage and ventilation of the sinus. Patients with chronic sinusitis may have bouts of acute sinusitis superimposed on chronic disease.

Pain is typically absent in patients with chronic sinusitis, except in those with an infected frontal sinus. In this latter group, most patients complain of headache that is a dull, constant ache. In patients with chronic sinusitis, the only symptoms may be nasal obstruction, postnasal drip, chronic cough, hyposmia, and unpleasant breath.

The relationship of sinusitis and asthma is unclear. Clinically confirmed sinusitis occurs in about 17% to 30% of patients with known asthma.[28,29] Approximately 12% of patients with chronic sinusitis have asthma, and this may be restricted to those with extensive sinusitis by computed tomography (CT).[30,31] An equal number of radiologic sinus abnormalities have been found in patients with asthma, regardless of whether their asthma was mild or severe.[29]

Several mechanisms are postulated for the relationship of sinusitis to asthma. It is possible that the pulmonary aspiration of infected sinus secretions could lead to a secondary bronchitis, resulting in asthma in susceptible patients. In a small study of patients with sinusitis

TABLE 1.—*Complications of Sinusitis*

Intracranial	*Extracranial*
Meningitis	Osteomyelitis
Epidural or subdural empyema	Orbital cellulitis
Brain abscess	Subperiosteal abscess
Cavernous sinus thrombosis	Orbital abscess
	Blindness
	Superior orbital fissure syndrome
	Epiphora

alone or sinusitis with asthma, there was no demonstration of the pulmonary aspiration of radionuclide-labeled maxillary sinus secretions.[32]

Sinusitis could also result in parasympathetic stimulation and reflex bronchospasm. Several, but not all, studies of animals and humans have shown that electrical, mechanical, or irritant-induced stimulation of the nasopharyngeal or sinus mucous membranes resulted in bronchoconstriction, as evidenced by increased airway resistance or a fall in pulmonary function.[33,34]

With the resolution of sinusitis, the clinical course of asthma may improve.[35] In one study, 50 adults with medically refractory sinusitis and asthma underwent a surgical procedure with an improvement or cure of their sinusitis.[36] All had symptomatic improvement in asthma, and most of the 28 patients on long-term corticosteroid therapy had a sustained reduction or discontinuation of the medication.

Complications of Sinusitis

The complications of sinusitis are listed in Table 1. Intracranial complications are more commonly seen with infection of the frontal and ethmoid sinus because of their proximity to the dura and drainage of the diploic veins from the frontal sinus into the dural veins. Sinusitis is the primary source of infection in as much as two thirds of patients with intracranial abscesses[37] and 5% of cases of community-acquired bacterial meningitis.[38]

Extracranial complications most often involve the orbit and occur more commonly in patients with ethmoid sinusitis because of the thinness of the orbital wall and its close proximity to the sinus and valveless ophthalmic venous system. These veins communicate between facial, sinus, orbital, and intracranial veins and drain directly into the cavernous sinus. Although orbital complications are seen more often in young children, adults and older children tend to have more severe involvement.[39]

Orbital complications can range from edema of the eyelids, orbital cellulitis, subperiosteal abscess with exophthalmos, and chemosis, to orbital abscess with pronounced exophthalmos, chemosis, and visual impairment, and finally, progression to cavernous sinus thrombosis. Blindness can result from these orbital complications.[40] Cavernous sinus thrombosis should be suspected in patients with bilateral ophthalmoplegia and loss of vision. The diagnosis of orbital complications is based on clinical and CT or magnetic resonance findings.

Epiphora may result from chronic inflammation of the nasal mucosa. It occurs due to either stenosis of the nasolacrimal duct or obstruction of its orifice in the inferior meatus. In patients with ethmoid sinusitis, inflammation may extend to the lacrimal sac and result in tearing.

The superior orbital fissure syndrome may occur in patients with acute or chronic sphenoid sinusitis. Clinically, there is palsy of the nervus abducens (VI) followed by involvement of the third, fourth, and fifth cranial nerves, orbital pain, exophthalmos, and ophthalmoplegia. Finally, recurrent attacks of sinusitis may result in osteomyelitis of the orbital plate or frontal or sphenoid bones.

Differential Diagnosis

Other causes of facial pain and headache that may mimic acute sinusitis include migraine and cluster headache, trigeminal neuralgia, pain originating from teeth, temporomandibular disorders, and temporal arteritis. It may be difficult to differentiate patients with acute sinusitis clinically from patients with protracted respiratory symptoms, both cough and nasal discharge, because these can also cause headache. Headache due to sinusitis, however, is aggravated by bending forward, coughing, or sneezing.

Diagnostic Studies

In primary care practice, acute sinusitis is generally a clinical diagnosis based on the history and physical examination findings. The paranasal sinuses may be visualized by standard radiography, including occipitomental (Waters'), occipitofrontal (Caldwell's), and lateral and oblique (Rhese) views.[41] A recent study suggested that a

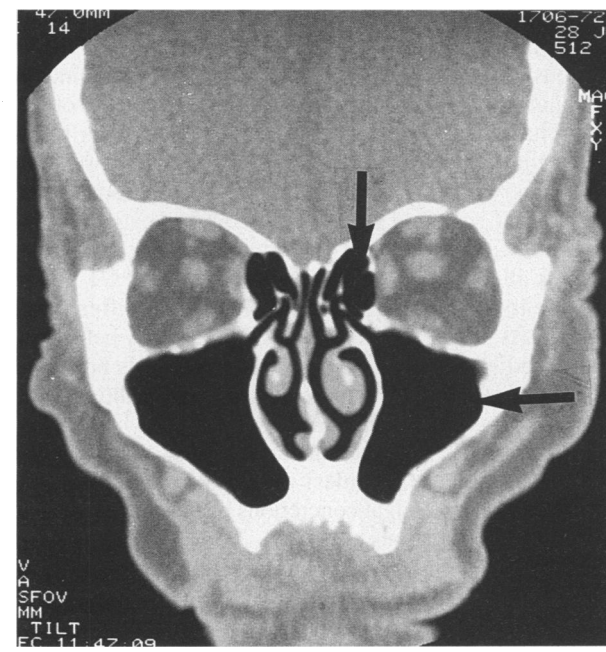

Figure 2.—A computed tomographic scan shows a normal maxillary sinus (**lower arrow**) and ethmoid sinus (**upper arrow**).

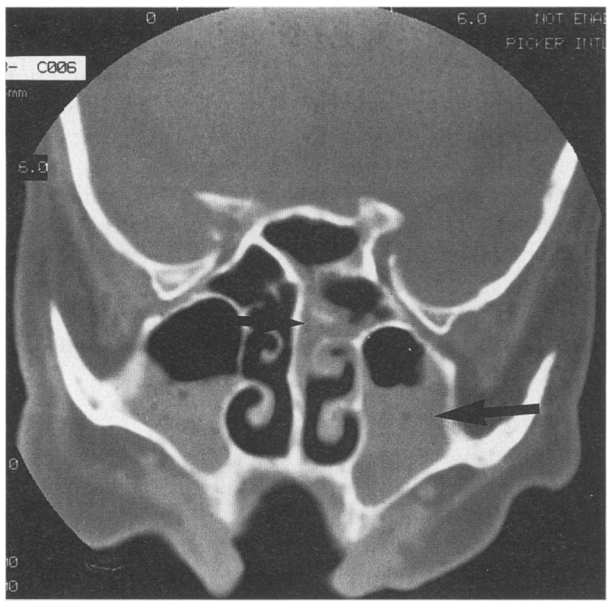

Figure 3.—A computed tomographic scan shows mucosal thickening and an air-fluid level in the left maxillary sinus (**lower arrow**) and mucosal thickening in the left ethmoid sinus (**upper arrow**) and left middle turbinate compared with normal turbinates on the opposite side.

single Waters' view was adequate for evaluating acute maxillary sinusitis in general medical patients with a high prevalence of sinusitis,[42] but it was not good for evaluating frontal, ethmoid, or sphenoid sinusitis. Because acute sinusitis is such a common and self-limited disorder, however, there is no consensus on how standard radiography should be used by primary care providers in the initial clinical evaluation of sinusitis.

Computed tomographic scanning shows excellent resolution of the paranasal sinuses, clarifying the anatomic relationships and variations that may play a role in sinusitis (Figures 2 and 3).[43] Recent studies evaluating the operating characteristics of CT scanning for sinusitis have shown that about 16% of persons without sinus-related symptoms will have abnormalities of the paranasal sinuses on CT scanning whereas only 62% of patients with sinus-related symptoms actually have CT scan evidence of sinus abnormalities.[44] The primary roles of CT scanning are to diagnose sinus disease when the differential diagnosis is not clear and to define the anatomy before an anticipated operation. Computed tomography is not used in the routine evaluation of acute sinusitis. Although the role of magnetic resonance imaging has yet to be defined in sinus disease, published articles suggest that it offers little in addition to CT, unless there is concern for a malignant neoplasm, fungal concretions of the sinuses, or intracranial complications.

More recently, diagnostic nasal endoscopy has been advocated as a primary evaluation strategy in those patients with recurrent or chronic symptoms.[45] This procedure, which can be done simply in the office by an otolaryngologist, provides a clear view of the anterior nasal cavity, including the middle meatus. Many now advocate that nasal endoscopy should supplement the diagnostic evaluation in all patients with chronic and recurrent acute sinusitis and that CT should be used either when endoscopy fails to explain the symptoms attributed to sinusitis or to define the anatomy before surgical therapy. In this regard, nasal endoscopy may serve as a screening test for deciding which patients require CT.

Medical Management

There are no proven methods to prevent acute sinusitis, although some authors recommend a prompt use of decongestants at the start of a viral upper respiratory tract infection in patients with a history of sinusitis. The treatment of conditions that may predispose to sinusitis, such as allergic rhinitis, nasal structural abnormalities, or dental root infection, may lessen the risk. Finally, adequate antimicrobial therapy for acute sinusitis, both in drug selection and duration, may help reduce the incidence of permanent mucosal damage and subsequent chronic sinusitis (Table 2).[7]

Timely adequate antibiotic coverage is the mainstay of the treatment of acute sinusitis, the goal being to relieve symptoms, prevent the development of acute complications, and avoid the development of chronic sinusitis.[46] Because there is no correlation between the organisms identified on nasopharyngeal culture and the causative agent found on sinus aspiration, antibiotic therapy is empirically directed at the most likely causative organism. Studies of sinus punctures before and after treatment have shown certain antimicrobial agents to be effective in cases of acute, community-acquired sinusitis, including combinations of trimethoprim and sulfonamides, aminopenicillins, and some cephalosporins, such as cefuroxime axetil.[3,14,46-49] No consensus exists about the duration of therapy. Although 10-day courses were adequate in these studies and many authors advocate 14-day regimens for the treatment of acute sinusitis,[50,51] a recent controlled trial showed equal efficacy between 3-day and 10-day regimens using trimethoprim and sulfamethoxazole.[52] In general, longer (10 to 14 days) duration therapy is standard practice.

An estimated 25% of *H influenzae* and *Moraxella catarrhalis* is β-lactamase–producing in cases of maxillary sinusitis in adults in some communities and, therefore, resistant to aminopenicillin drugs.[51,53] The combination of trimethoprim-sulfamethoxazole provides coverage (is bactericidal) against these β-lactamase–producing organisms, and generic products may even be less expensive than the aminopenicillins.[53,54] Agents such as cefuroxime axetil are effective against these resistant organisms, as well as *S aureus*. The combination of amoxicillin and clavulanate potassium provides a similar broad-spectrum bactericidal coverage in acute sinusitis and has been effective clinically and in sinus puncture studies.[55-57] All of these agents are first-line choices for the treatment of acute sinusitis, with trimethoprim-sulfamethoxazole being the agent with the broadest coverage for the least expense (see Table 2) and

TABLE 2.—*Management of Sinusitis*

Type of Sinusitis	Therapy		Usual Adult Dosage, mg orally/day	Price, $*
Acute sinusitis				
Community-acquired.....	Antibiotic			
	Primary:	Trimethoprim-sulfamethoxazole, DS†	160-800, 2×	2.28
		Amoxicillin‡	500, 4×	10.92
	Alternate§:	Amoxicillin-clavulanate potassium	500-125, 3×	59.00
		Cefuroxime axetil	250, 2×	52.00
		Cefixime	500, 1×	50.00
		Clarithromycin	500, 2×	55.00
	Adjunct			
	Intranasal‖ or oral decongestant			
	Seek urgent otolaryngology referral for complications of sinusitis			
	Seek elective otolaryngology referral for >3 episodes/yr			
Hospital-acquired	Antibiotic			
	Primary:	Imipenem		
		3rd-generation parenteral cephalosporin		
		Either β-lactamase–resistant penicillin, ticarcillin disodium-clavulanate potassium, or piperacillin sodium-tazobactam sodium with an antipseudomonal aminoglycoside		
	Alternate:	Substitute aztreonam for the antipseudomonal aminoglycoside coverage listed above		
		Fluoroquinolone and ampicillin		
	Adjunct			
	Remove foreign body if associated (nasogastric or nasotracheal tube, packing)			
	Seek urgent otolaryngology referral			
Special groups				
Immunocompromised—HIV-positive, neutropenic, diabetic ketoacidosis	Antibiotic			
	Primary:	Coverage for gram-negative bacteria and opportunistic fungi		
	Adjunct			
	Seek urgent otolaryngology referral			
Allergic sinusitis.....	Antibiotic			
	Primary:	Treat acute exacerbations the same as community-acquired acute sinusitis		
	Adjunct			
	Decongestant‖			
	?Topical corticosteroid			
	?Antihistamine			
Chronic sinusitis	Antibiotic			
	Primary:	Treat acute exacerbations the same as community-acquired acute sinusitis		
	Adjunct			
	Decongestant‖			
	?Topical corticosteroid			

DS = double strength, HIV = human immunodeficiency virus

*Average wholesale price for a 10-day supply.

†Not effective in sinusitis following dental procedures where anaerobes may be present.

‡Aminopenicillins should be used for first-line therapy only where community prevalence of β-lactamase–resistant *Haemophilus influenzae* is low.

§Alternates are equally efficacious, but more expensive; if treatment response is incomplete after a 10- to 14-day regimen with a primary antibiotic, a 14- to 21-day course of an alternate antibiotic is recommended.

‖To prevent a rebound phenomenon, intranasal decongestants should not be used for more than 7 days.

aminopenicillins being used in sulfa-allergic patients in areas with a low prevalence of antibiotic resistance. Cefuroxime axetil, cefixime, and amoxicillin-clavulanate are considerably more expensive alternatives. New macrolide antibiotics, such as clarithromycin, may also be efficacious as first-line agents.[58,59]

We advocate using trimethoprim-sulfamethoxazole for initial treatment, unless the patient is allergic to sulfa or the sinusitis occurs after a dental procedure; amoxicillin or ampicillin should be used only if β-lactamase–producing organisms are uncommon, as determined by sensitivity testing in the locale (Table 2). If there is only partial clinical resolution after 10 to 14 days of taking a first-line antibiotic, a second course of treatment using a β-lactamase–resistant drug such as amoxicillin-clavulanate or a second- or third-generation cephalosporin should be initiated. Although some authors advocate confirming the diagnosis with plain radiography (or CT) after treatment fails with a first-line agent,[46] further study is needed to clarify whether imaging at this point alters outcomes.[60]

The antral sinus should be aspirated to detect causative organisms in patients with hospital-acquired sinusitis and in immunocompromised patients because of the increased prevalence of fungal, gram-negative, and polymicrobial infections. In patients with nosocomial infection and while awaiting culture results, or if treating empirically, the use of a β-lactamase–resistant antipseudomonal penicillin such as piperacillin, mezlocillin, or ticarcillin disodium and clavulanate potassium or piperacillin sodium and tazobactam sodium, in combination with an antipseudomonal aminoglycoside, is recommended. Other first-line options include a third-generation cephalosporin with enhanced antipseudomonal activity, such as ceftazidime or cefoperazone sodium, with or without an additional antipseudomonal aminoglycoside, or imipenem. Alternative drug regimens include a fluoroquinolone antimicrobial agent and ampicillin or substituting aztreonam for the aminoglycoside in the previous regimens.[53] Nondrug therapy includes the removal of any obstructing foreign body (such as a nasogastric or nasotracheal tube) and referral to an otolaryngologist for therapeutic antral lavage or surgical drainage.

In addition to using antimicrobial agents, the treatment of acute sinusitis includes facilitating adequate drainage by reducing tissue edema and maintaining ostial patency. Adjuncts to antimicrobial therapy that have been reported anecdotally include pharmacologic measures such as oral and topical decongestants, antihistamines, topical corticosteroids, expectorants, and analgesics. Nonpharmacologic measures include saline sprays and steam inhalations. A paucity of scientific data is available to support most of these adjuncts, however, and their use has been based on theoretical considerations and favorable clinical impressions. The average patient with acute sinusitis will have rapid subjective improvement within the first five days of treatment that precedes radiographic improvement.[61]

Oral vasoconstrictors, known as decongestants, that are available over-the-counter or as a prescription, are pseudoephedrine, phenylpropanolamine, and phenylephrine. These agents are sympathomimetic, α-adrenergic agonists that reduce nasal blood flow. Because they are parenteral oral decongestants, they have the theoretical advantage over topical agents to act on tissues deep within the osteomeatal complex to decrease tissue congestion and facilitate drainage. In patients with chronic sinusitis, a single 100-mg dose of phenylpropanolamine decreased nasal airway resistance and increased the functional osteomeatal size.[62] Although concerns have been raised about potentiating hypertension in patients taking oral decongestants, a number of studies have failed to demonstrate clinically important changes in heart rate or blood pressure in normotensive or hypertensive subjects.[63-65]

Topical (intranasal) decongestants have also been shown to increase nasal airway patency in patients with rhinitis, which act locally to decrease nasal edema and facilitate sinus drainage through the ostia.[66] Nasal sprays such as 0.5% phenylephrine hydrochloride (HCl) and 0.05% oxymetazoline HCl may provide immediate symptomatic relief. Other common intranasal decongestants are naphazoline HCl, tetrahydrozoline HCl, and xylometazoline HCl. Intranasal decongestants should be administered with the head erect, one to three sprays in each nostril, two to three times a day for three to seven days. Prolonged use may cause rebound vasodilatation, called rhinitis medicamentosa, which may result from the inhibition of α-adrenergic receptors due to high local concentrations of the drug, chronic chemical irritation, and reactive hyperemia.[51,67,68]

No studies have been done to show whether or not antihistamines are beneficial in patients with acute or chronic sinusitis. Antihistamines have both anticholinergic and decongestant properties to inhibit secretion and, therefore, are relatively contraindicated in patients with acute sinusitis; otolaryngologists do not recommend using antihistamines in this situation due to concerns of the drying of mucous membranes and interference with the clearing of secretions.[50]

Topical corticosteroids have been reported to reduce local sinus ostial inflammation and so increase functional ostial diameter, but there are no adequate data to justify their use in all patients with sinusitis. Many advocate the use of topical corticosteroids for patients with allergic rhinitis or sinusitis or in patients with chronic sinusitis.[50,69] In a recent placebo-controlled, double-blind study of the use of intranasal steroids in an adult population recruited from allergy clinics, their use with a broad-spectrum antibiotic in acute sinusitis had limited efficacy; there was only a trend toward lessening symptoms and decreasing inflammation.[70]

Expectorants such as guaifenesin are used by some in an attempt to thin secretions and aid in drainage, although there are no data related to their use in sinusitis. Analgesics are used purely as symptom control in those patients with substantial facial pain or headache. The use of nonpharmacologic measures is not supported by data, but many of these methods seem to provide symptomatic relief.[50,69] They include steam inhalation to liquefy secretions and moisturize dry, inflamed mucous membranes. Topical saline spray or irrigation with a saline solution two or three times a day may provide a mild decongestant

TABLE 3.—*Indications for Referral to an Otolaryngologist**

Acute Sinusitis
Complications
Deterioration within 2 days
Treatment failure after 2 courses of appropriate antibiotics
Frequent recurrences (>3 episodes/year)
Nosocomial infections
Immunocompromised hosts
Chronic Sinusitis
Treatment failure (or medically refractory)
Persistent nasal polyps with nasal obstruction

*See text for discussion.

effect, liquefy secretions, and moisturize the mucous membranes while providing some short-term symptomatic relief.

Chronic sinusitis is a disease of mucosal damage and is not primarily an infectious state.[71-73] Acute infectious exacerbations of chronic sinusitis should be treated similarly to acute sinusitis, but for treatment durations of three or more weeks.[27,51] Some authors advocate treatment in these situations with antibiotics such as penicillin, ampicillin, clindamycin, or amoxicillin-clavulanate to eradicate anaerobes as well as the major organism seen with acute exacerbation.[53] In addition to treatment with antibiotics for acute exacerbations, the use of decongestants and topical corticosteroids may aid in reducing the inflammatory response in patients with both acute flare and chronic infection.[74-76] A multimodal approach has been advocated, including administering antibiotics for 21 days and beclomethasone nasal spray, guaifenesin-pseudoephedrine HCl tablets, steam inhalations, and nasal saline sprays for 30 days.[27] Sinus lavage may be required to identify the organisms involved for recurring infections, but ultimately surgical intervention is often required to facilitate sinus drainage.

Referral to an Otolaryngologist

Table 3 lists the indications for referring patients with sinusitis to an otolaryngologist who can do nasal endoscopy and an antral puncture to obtain material for culture and to facilitate therapeutic irrigation. All of these patients will be evaluated by sinus CT series.

The primary surgical approach for sinusitis has shifted from the radical resection of damaged mucosa to endoscopic operations for correcting obstruction of the osteomeatal complex, allowing the sinus mucosa to heal with adequate ventilation and drainage, and thus restoring mucociliary function.[77] Indications for functional endoscopic sinus operations include medically refractory chronic sinusitis and recurrent acute sinusitis associated with abnormalities of the osteomeatal complex.[5,78,79] Structural abnormalities that can be detected include mucoceles, sinus or nasal polyps, and concha bullosa of the middle turbinate that obstructs the osteomeatal complex. Only if symptoms persist despite medical therapy and correlate with objective endoscopic and CT scan findings is surgical therapy warranted. In a group of patients with chronic sinusitis with a median one-year follow-up, 90% felt that surgical therapy lessened symptoms, with 50% having complete resolution of their symptoms.[80] Results were better in those patients in whom a substantial nasal deformity was corrected, and relief of facial pain was more common than the resolution of other symptoms. An opacified sphenoid sinus portended a poor outcome. Of this group of 155 patients, 11% required a second endoscopic operation, and 6% later required traditional surgical treatment.

In summary, community-acquired sinusitis in adults is a common problem. Diagnosis generally rests on historical and examination features. Transillumination and plain radiography are rarely used by clinicians any longer. Most patients can be managed successfully with a regimen of empiric antibiotics and oxymetazoline nasal decongestant for 10 to 14 days. Because pneumococci and *H influenzae* are the most common causal organisms, trimethoprim-sulfamethoxazole or amoxicillin-clavulanate are first-line antibiotic choices. A lack of resolution or the frequent recurrence of sinusitis despite longer-term therapy with a penicillinase-resistant antibiotic warrants evaluation with CT imaging and specialty referral.

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